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SYSTEM:OS  - DIALOG OneSearch
  File  55:BIOSIS PREVIEWS(R)  1985-1995/May W4
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  File 399:CA SEARCH(R)  1967-1995/UD=12222
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      Set  Items  Description
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?s vla? and sclerosis

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          48964  SCLEROSIS
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?rd s1

...completed examining records
      S2    12  RD S1 (unique items)
?t s2/7/all


 2/7/1     (Item 1 from file: 55)
DIALOG(R)File  55:BIOSIS PREVIEWS(R)
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11025442     BIOSIS Number: 97225442
  Retroviral synthetic peptide serum antibodies in human sporadic
amyotrophic lateral sclerosis
  Westarp M E; Foerhring B; Rasmussen H; Schraff S; Mertens T; Kornhuber H
H
  Ulm Univ. Dep. Neurology, Prittwitzstr. 43, W-89070 Ulm, GER
  Peptides (Tarrytown) 15 (2). 1994.  207-214.
  Full Journal Title:  Peptides (Tarrytown)
  ISSN: 0196-9781
  Language: ENGLISH
  Print Number: Biological Abstracts Vol. 097 Iss. 010 Ref. 142558
  We examined 101 sera from 32 adult sporadic amyotrophic lateral sclerosis
(ALS)  patients,  including  nine with positive enzyme-linked immunosorbent
assay (ELISA) serum antibodies against human spuma retrovirus (HSRV) (human
foamy virus (HFV)) envelope (env) and/or capsid (gag) proteins, for peptide
seroreactivity.  Synthetic  peptides  10  to  14 amino acids in length were
selected  from  HSRV  (3),  maedi-visna  virus  (1),  human  nerve  growth
factor-beta  (1), and human amyloid-beta sequences (1). Eighteen of 101 ALS
sera  compared with six of 144 control sera reacted to any of the sequences
(p  lt  0.01)  (i.e., 8/32 ALS patients and 2/93 control patients bound to a
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synthetic peptide, p lt 0.01). Peptide VLA- (NGF-beta(1-(4)) was reproducibly recognized by one of the 93 neurologic controls. and one of the 32 ALS patients reproducibly reacted to synthetic peptides (EET-, HSRV-env/NGF-beta(55-61)) and (GSN-, beta-amyloid(25-35)) simultaneously. This amyloid-A(25-35) peptide corresponds to the neurotoxic and neurotrophic tachykinin homology sequence described by Yankner. Only ALS patients (no controls) reacted with the visna/CNTF peptide SMC- and HSRV-bel-1/amyloid(740-751) peptide EGP-. Testing a total of 245 sera from 125 patients, three reproducible reactivities (two ALS, one OND) were observed both with and without glutaraldehyde linkage. Of the four peptides recognized either by more than one serum from the same patient with ALS or by sera from ALS patients only (EET-, GSN-, SMC-, EGP-), two share a circumscript homology with maedi-visna virus envelope glycoprotein (Table 1). Either retrovirally altered gene products or retroviral fragments could lead to a chronic neuronotrophic signal interference in sporadic ALS. As in murine models, such a retroviral involvement need neither imply an infection of motor neurons nor an autoimmune association between immunology and pathophysiology.

2/7/2 (Item 2 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

10992758 BIOSIS Number: 97192758
Expression of cell adhesion molecules associated with germinal center in Hodgkin's disease: An immunohistochemical study: The germinal center related complex and histologic subtypes
Ree H J; Khan A A; Qureshi M N; Teplitz C
Dep. Pathol., Beth Israel Med. Cent., First Ave. 16th Street, New York, NY 10003, USA
Cancer (Philadelphia) 73 (4). 1994. 1257-1263.
Full Journal Title: Cancer (Philadelphia)
ISSN: 0008-543X
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 009 Ref. 126426
Background. Of the four elements known to be associated with germinal center formation-B-cells, follicular dendritic cells, T-cells, and the cell adhesion molecules-the first three have been well studied in Hodgkin's disease, especially the nodular lymphocytic predominance subtype, established as a tumor of germinal center origin. However, no study has been done on the expression of the cell adhesion molecules associated with germinal center formation in Hodgkin's disease. Methods. Using the avidin-biodin peroxidase complex method, we studied the staining patterns for CD11a (lymphocyte function-associated antigen 1), CD54 (intercellular cell adhesion molecule 1), and very late antigen 4 (VLA-4) in frozen sections from 24 cases of Hodgkin's disease, along with those for follicular dendritic cell and cell surface markers for lymphocytes. Results. Reed-Sternberg cells and their variants and histiocytic cells stained for CD54. Lymphocytes stained for CD11a. Lymphocytes either formed patchy aggregates or dispersed without forming aggregates. Aggregating lymphocytes expressed CD20 (L-26), whereas dispersed, nonaggregating lymphocytes expressed CD3/CD4, or CD3/CD8. Extracellular matrices of these CD20+ B-cell aggregates stained for CD54, VLA-4, and follicular dendritic cells. CD54 staining revealed four patterns of reaction products deposits: discretely patchy, confluent, predominantly diffuse, and diffuse only. The discrete-patch predominance pattern was seen in the lymphocytic predominance type, both nodular (n = 3) and diffuse (n = 2), and in classic

nodular sclerosis with broad collagen bands (n = 4). The confluent pattern was seen in tumors with features of cellular-phase nodular sclerosis versus mixed cellularity type (n = 3). The predominantly diffuse was observed in the remainder of nodular sclerosis type with infrequent, narrow collagen bands (n = 5), in cellular-phase nodular sclerosis (n = 1), in cellular-phase nodular sclerosis versus mixed cellularity type (n = 2), and in mixed cellularity (n = 2). The diffuse-only pattern occurred in mixed cellularity with abundant fibrohistiocytoid stromal cells (n = 2). Conclusions. The cell adhesion molecules associated with germinal center formation were expressed in the great majority of cases of Hodgkin's disease. The expression was closely associated with the occurrence of distinctive CD20+ B-cell aggregates and follicular dendritic cell networks, forming a germinal center-related complex, and the presence of the complex correlated with nodular sclerosing features.

2/7/3 (Item 3 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

10539214 BIOSIS Number: 96139214
BINDING OF AN SJL T CELL CLONE SPECIFIC FOR MYELIN BASIC PROTEIN TO SJL BRAIN MICROVESSEL ENDOTHELIAL CELLS IS INHIBITED ANTI-VLA-4 OR ITS LIGAND ANTI-VASCULAR CELL ADHESION MOLECULE 1 ANTIBODY
TANAKA M; SATO A; MAKINO M; TABIRA T
DEP. NEUROLOGY, BRAIN RES. INST., NIIGATA UNIV., NIIGATA 951, JPN.
J NEUROIMMUNOL 46 (1-2). 1993. 253-257. CODEN: JNRID
Full Journal Title: Journal of Neuroimmunology
Language: ENGLISH
Adhesion molecules probably are required for the migration of T lymphocytes to inflamed tissues, but the roles of these molecules have yet to be understood in the pathogenesis of inflammatory diseases such as multiple sclerosis. The adhesion of an SJL murine T cell clone specific for myelin basic protein (MBP) to endothelial cells (ECs) from SJL newborn brain microvessels was examined. Sixty percent of the 2 .times. 104 T cell clones stimulated once every 2 weeks with MBP were bound to ECs, whereas less than 5 of the same number of lymphocytes from peripheral lymph nodes were bound. However, binding was not central nervous system (CNS)-specific . Monoclonal antibody to VLA-4 or VCAM-1 partially inhibited the binding of the T cell clone to ECs. Binding of the T cell clone to ECs increased when the latter were incubated with IL-1 or TNF, but was not inhibited by anti-VLA-4 or VCAM-1. We suggest that the VLA-4/VCAM-1 pathway functions in the binding of the T cell clone specific for MBP to brain ECs but that adhesion molecules other than VLA-/VCAM-1 are involved because anti-VLA-4 and anti-VCAM-1 did not produce complete inhibition.

2/7/4 (Item 4 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

10502346 BIOSIS Number: 96102346
ADHESION MOLECULE EXPRESSION ON CEREBROSPINAL FLUID T LYMPHOCYTES EVIDENCE FOR COMMON RECRUITMENT MECHANISMS IN MULTIPLE SCLEROSIS ASEPTIC MENINGITIS AND NORMAL CONTROLS
SVENNINGSSON A; HANSSON G K; ANDERSEN O; ANDERSSON R; PATARROYO M; STEMME S
DEP. CLIN. CHEM., SAHLGREN'S HOSP., S-413 45 GOTHENBURG, SWED.

ANN NEUROL 34 (2). 1993. 155-161. CODEN: ANNED
Full Journal Title: Annals of Neurology
Language: ENGLISH

The expression of T-cell surface antigens was investigated in the cerebrospinal fluid (CSF) and peripheral blood of 11 patients with multiple sclerosis, 6 patients with aseptic meningitis, and 16 healthy subjects. A panel of monoclonal antibodies to adhesion and activation proteins was used in combination with an anti-CD3 antibody in dual-color flow cyotmetry. The problem of low cell numbers in the CSF from normal individuals was overcome by use of a modified staining procedure in microtiter plates, enabling analysis of as few as 5,000 cells. The majority of T cells in the CSF of the three patient groups exhibited the phenotype of memory cells (CD45RO+). CSF T cells also expressed significantly higher levels of several adhesion and activation molecules, including very late activation (VLA) antigens 3 through 6, lymphocyte function-associated (LFA) antigen, 1, LFA-3, CD2, CD26, and CD44. Comparison between the different categories revealed that peripheral blood T cells from patients with multiple sclerosis expressed significantly lower amounts of the VLA integrins 4 and 5 as well as their common .beta. subunit CD29, compared with normal control subjects. No differences between patients with multiple sclerosis and control subjects could, however, be seen regarding the distribution of memory/naive cells or CD4+/CD8+ cells in peripheral blood. Our data support a hypothesis that memory T cells with a high expression of several adhesion moelcules are selectively recruited to the central nervous system compartment, under both pathological and normal conditions. We also provide evidence for an altered expression of adhesion molecules on peripheral blood T cells in patients with multiple sclerosis that is independent of the memory cell phenotype as defined by the expression of the CD45RO epitope.

2/7/5 (Item 5 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

10417044 BIOSIS Number: 96017044
DUAL EXPRESSION OF CD45RA AND CD45RO ISOFORMS ON MYELIN BASIC PROTEIN-SPECIFIC CD4-POSITIVE T-CELL LINES IN MULTIPLE SCLEROSIS
QIN Y; VAN DEN NOORT S; KURT J; GUPTA S
MED. SCI. I, C-264A, UNIV. CALIFORNIA, IRVINE, CALIFORNIA 92717, USA.
J CLIN IMMUNOL 13 (2). 1993. 152-161. CODEN: JCIMD
Full Journal Title: Journal of Clinical Immunology
Language: ENGLISH

Myelin basic protein (MBP)-specific T-cell lines from patients with multiple sclerosis (MS) and healthy controls were analyzed for the expression of CD45 isoforms and adhesion molecules. In the multiple sclerosis group, 22 of 24 MBP-specific T-cell lines were CD4+. Two distinct patterns were observed with regard to CD45 isoform expression. Pattern I showed dual expression of CD45 isoforms (CD4+ CD45RA+ CD45RO+ CD29+) and Pattern II included cells with a single CD45 isoform (CD4+ CD45RA-CD45RO+CD29+). All 10 cell lines from healthy controls were CD4+ and displayed Pattern II (CD4+ CD45RA-CD45RO+CD29+). The dual expression of CD45 isoform in T-cell lines from MS was stable, did not represent a transition stage from CD45RA to CD45RO, and was cell-cycle independent. All cell lines from MS and controls expressed increased levels of LFA-1 (CD11a), LFA-2 (CD2), LFA-3 (CD58), ICAM-1 (CD54), and VLA-4 (CDw49d). These data show the presence of unique MBP-specific T cells (CD4+ CD45RA+CD45RO+CD29+) that might play a role in the pathogenesis of MS.

2/7/6 (Item 6 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

9617209 BIOSIS Number: 94122209
ADHESION RECEPTOR PROFILE OF THYMIC B-CELL LYMPHOMA
EICHELMANN A; KORETZ K; MECHTERSHEIMER G; MOELLER P
PATHOLOGISCHES INSTITUT, UNIVERSITAET HEIDELBERG, IM NEUENHEIMER FELD 220, D-6900 HEIDELBERG, GERMANY.
AM J PATHOL 141 (3). 1992. 729-741. CODEN: AJPAA
Full Journal Title: American Journal of Pathology
Language: ENGLISH
Primary thymic B-cell lymphoma is clinically characterized by aleukemic, highly aggressive local growth, infrequent distant metastasis, and infrequent secondary lymph node involvement. VLA-1 to VLA-6 are cell surface molecules binding to matrix molecules such as collagen, fibronectin, epiligrin, and laminin. VLA-4 additionally binds to VCAM-1 and ICAM-2, thus mediating intercellular adhesion. Other molecules involved in cell/cell adhesion are LFA-1 (CD11a/CD18), Mac-1(CD11b/CD18) and their ligand ICAM-1 (CD54), p150,95 (CD11c/CD18), LFA-3 (CD58), CD44, and LECAM-1. Twenty-three tumors, together with normal lymphoid tissue, were immunohistochemically examined to investigate the expression pattern of these molecules in thymic B-cell lymphomas and in their putative normal counterparts, namely thymic medullary B cells. Thymic B-cell lymphomas consistently lacked VLA-1,-2,-3,-5,-6, and CD11b, expressed ICAM-1 in 21 of 23 cases but were heterogenous for VLA-4, LFA-1, CD11c, LFA-3, CD44, and LECAM-1. Presence of LFA-1 correlated with LFA-3 expression (P = 0.029). The receptor profile of thymic B-cell lymphoma was reminiscent of the expressional status of normal thymic medullary B cells in some aspects but deviated in others: Assuming that, in terms of differentiation, thymic B-cell lymphoma is related to the asteroid variant of thymic medullary B cells, a propensity to down-regulate/lose VLA-4, CD11a, CD44, and LECAM-1 would have to be supposed in conjunction with a tendency to overexpress ICAM-1 and LFA-3. Sclerosis as an inconsistent phenomenon in thymic B-cell lymphoma was absent in 8 of 23 tumors. Presence of sclerosis correlated with LECAM-1 expression of the tumor cells (P = 0.038). Recent studies suggest that a locally growing/aleukemic phenotype of a B-cell neoplasia might be determined by the phenotype VLAs-, LFA-1+, ICAM-1+, CD44-, and LECAM-1-. Our data corroborate this view.

2/7/7 (Item 7 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

8896567 BIOSIS Number: 42121567
VLA ALPHA-1-ALPHA-6 AND ALPHA-V INTEGRINS IN GLOMERULONEPHRITIS
CUZIC S; WALDHERR R
DEP. PATHOL., SVEUCILISNA BOLNICA, ZAGREB, YUGOSL.
SYMPOSIUM ON MESANGIAL CELLS AND EXTRACELLULAR MATRIX, ERLANGEN-NUERNBERG, GERMANY, JUNE 9-12, 1991. KIDNEY INT 41 (3). 1992. 701. CODEN: KDYIA
Language: ENGLISH

2/7/8 (Item 1 from file: 72)
DIALOG(R)File 72:EMBASE

9588716 EMBASE No: 95158342
T lymphocyte adhesion to human brain pericytes is mediated via very late antigen-4/vascular cell adhesion molecule-1 interactions
Verbeek M.M.; Westphal J.R.; Ruiter D.J.; De Waal R.M.W.
Department of Pathology, University Hospital Nijmegen, P.O. Box 9101, 6500 HB Nijmegen Netherlands
Journal of Immunology (USA) , 1995, 154/11 (5876-5884) CODEN: JOIMA ISSN: 0022-1767
LANGUAGES: English SUMMARY LANGUAGES: English
T cell adhesion to the brain microvascular endothelium and subsequent migration into the brain parenchyma is one of the major events in the development of multiple sclerosis (MS). Interactions of the T cell integrin very late antigen-4 (VLA-4) with its receptor, vascular cell adhesion molecule-1 (VCAM-1) have been described to be of crucial importance for the development of MS. We investigated the expression of these adhesion molecules in MS brain tissue by immunohistochemical analysis, and studied their functional involvement in an in vitro T cell adhesion assay. A number of other adhesion molecules were studied for comparison. In cryosections of several MS brains, expression of VCAM-1 was demonstrated not only on the endothelium of vessels surrounding MS plaques, but also in perivascular positions, suggesting expression by pericytes. T cells adhered to both cell types in vitro. Both LFA-1/intercellular adhesion molecule-1 and VLA-4/VCAM- 1 interactions were equally involved in the adhesion of T cells to TNF-alpha- stimulated endothelial cells. However, adhesion of T cells to TNF-alpha- stimulated pericytes was clearly dominated by VLA-4/VCAM-1 interactions. These results indicate that pericytes, next to endothelial cells, may play an important role in regulating T cell infiltration into the central nervous system.

2/7/9 (Item 2 from file: 72)
DIALOG(R)File 72:EMBASE

9581612 EMBASE No: 95152539
Basic aspects of neuroimmunology as they relate to immunotherapeutic targets: Present and future prospects
Dalakas M.C.
Medical Neurology Branch, NINDS, NIH, 10 Center Drive, Bethesda, MD 20892-1382 USA
Annals of Neurology (USA) , 1995, 37/SUPPL. 1 (S2-S13) CODEN: ANNED ISSN: 0364-5134
LANGUAGES: English SUMMARY LANGUAGES: English
The neurological diseases with definite or putative immune pathogenesis include myasthenia gravis; Lambert-Eaton myasthenic syndrome; IgM monoclonal anti-myelin-associated glycoprotein-associated demyelinating polyneuropathy; Guillain-Barre syndrome; chronic inflammatory demyelinating polyneuropathy; multifocal motor neuropathy with or without GM1 antibodies; multiple sclerosis; inflammatory myopathies; stiff-man syndrome; autoimmune neuromyotonia; paraneoplastic neuronopathies and cerebellar degeneration; and neurological diseases associated with systemic autoimmune conditions, vasculitis, or viral infections. The events that lead to these autoimmune diseases are not clear but the following sequential steps are critical: (a) the breaking of tolerance, a process in which cytokines, molecular mimicry, or superantigens may play a role in rendering previously anergic T cells to recognize neural autoantigens; (b) antigen recognition by the T-cell

receptor complex and processing of the antigen via the major histocompatibility complex class I or II; (c) costimulatory factors especially B7 and B7- binding proteins (CD28, CTLA-4) and intercellular adhesion molecule (ICAM)-1 and its leukocyte function-associated (LFA)-1 ligand; (d) traffic of the activated T cells across the blood-brain or blood-nerve barrier via a series of adhesion molecules that include selectins, leukocyte integrins (LFA-1, Mac-1, very late activating antigen (VLA)-4) and their counterreceptors (ICAM-1, vascular cell adhesion molecule (VCAM)) on the endothelial cells; and (e) tissue injury when the activated T cells, macrophages, or specific autoantibodies find their antigenic targets on glial cells, myelin, axon, calcium channels, or muscle. In designing specific immunotherapy, the main players involved in every step of the immune response need to be considered. Targets for specific therapy in neurological diseases include agents that (a) interfere or compete with antigen recognition or stimulation, (b) inhibit costimulatory signals or cytokines, (c) inhibit the traffic of the activated cells to tissues, and (d) intervene at the antigen recognition sites in the targeted organ. The various immunomodulating procedures and immunosuppressive drugs currently used for nonselective neuroimmunotherapy are discussed in the context of their interference with the above-described immune mediators.

2/7/10 (Item 3 from file: 72)
DIALOG(R)File 72:EMBASE

9546630 EMBASE No: 95117826
The adhesion molecule and cytokine profile of multiple sclerosis lesions
Cannella B.; Raine C.S.
Department of Pathology, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY 10461 USA
Annals of Neurology (USA) , 1995, 37/4 (424-435) CODEN: ANNED ISSN: 0364-5134
LANGUAGES: English SUMMARY LANGUAGES: English
The expression of the adhesion molecules, vascular cell adhesion molecule- 1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1), and their respective receptors on leukocytes, very late activation antigen-4 (VLA-4) and lymphocyte function-associated antigen-1 (LFA-1), together with a selection of proinflammatory and immunomodulatory cytokines (interleukin (IL)-1, IL-2, IL-4, IL-10, tumor necrosis factor-alpha (TNF-alpha), transforming growth factor-beta (TGF-beta), and interferon-gamma (IFN-gamma)) was examined by immunocytochemistry in multiple sclerosis (MS) lesions of different ages and compared with central nervous system (CNS) tissue from other neurological diseases, both inflammatory and noninflammatory, and normal CNS tissue. These molecules play key roles in lymphocytic infiltration and interactions during tissue inflammation and are in large part normally not expressed by CNS cells. High levels of expression of all the molecules tested were found in MS, particularly in chronic active lesions. Positivity for all molecules was also seen in other neurological diseases, even in noninflammatory conditions. There was some suggestion that the VCAM-1/VLA-4 adhesion pathway was expressed at higher levels in chronic MS lesions, while ICAM-1/LFA-1 was used more uniformly in lesions of all ages. Of the cytokines examined, there was increased expression of TNF-alpha and IL-4 in MS; this was found to be statistically significant when compared with noninflammatory neurological diseases. The expression of most adhesion molecules and some cytokines was negligible in normal CNS tissue although low-level reactivity for ICAM-1 TGF- beta, IL-4,

TNF-alpha, and IL-10 was detected, perhaps indicative of immunoregulatory mechanisms. Microglial cells and astrocytes were the major CNS cell types expressing cytokines. The results indicate a potential in the CNS for widespread induced expression of molecules involved in the inflammatory cascade. No adhesion or cytokine molecule or pattern of expression unusual for MS was apparent.

2/7/11 (Item 4 from file: 72)
DIALOG(R)File 72:EMBASE

8639214 EMBASE No: 92319708
Lymphocyte migration into the CNS modelled in vitro
Male D.; Pryce G.; Linke A.; Rahman J.
Department of Neuropathology, Institute of Psychiatry, Denmark Hill, London SE5 8AF United Kingdom
J. NEUROIMMUNOL. (Netherlands) , 1992, 40/2-3 (167-171) CODEN: JNRID ISSN: 0165-5728
LANGUAGES: English SUMMARY LANGUAGES: English
We report on a series of experiments which examines the factors controlling lymphocyte adhesion to brain endothelium in vitro and the factors which control cell migration across the endothelium, using a new migration assay. Although lymphocyte adhesion preceded migration across the brain endothelium, the two processes are not identical. We noted that activated CD4+ T cells were particularly good at migrating across endothelia. CD8+ T cells and B cells did not migrate but adhered well to endothelia. Moreover, the endothelium maintained high levels of cell traffic without being disrupted and without exhausting the molecular systems which allowed migration. From the viewpoint of migration of dividing cells, the state of lymphocyte activation appeared to be the most important controlling factor - these cells migrated equally well across endothelium activated with cytokines or untreated endothelium. The kinetics of adhesion suggested that the LFA-1/ICAM-1 and VLA-4/VCAM combinations of adhesion molecules were important in controlling migration. With antibody blocking studies, the role of the LFA-1/ICAM-1 system was equivocal. While anti-LFA-1 blocked lymphocyte adhesion, anti-ICAM-1 did not, suggesting that the level of ICAM-1 was not critical.

2/7/12 (Item 1 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

09159161 95089161
[Adhesion molecules and immune-mediated diseases of the central nervous system]
Mizobuchi M; Iwasaki Y
Department of Neurological Sciences, Tohoku University School of Medicine.
Nippon Rinsho (JAPAN) Nov 1994, 52 (11) p2830-6, ISSN 0047-1852 Journal Code: KIM
Languages: JAPANESE Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL English Abstract
Expression of adhesion molecules in immune-mediated diseases of the central nervous system was reviewed. In multiple sclerosis and experimental allergic encephalomyelitis (EAE), endothelial cells of active lesions

increase expression of the adhesion molecules such as ICAM-1, VCAM-1 and inflammatory cells including memory T cells and macrophages express high levels of adhesion molecules such as LFA-1, VLA-4. Astrocytes also express CD44, ICAM-1, VCAM-1 and E-selectin in response to cytokine stimuli. In EAE, the majority of infiltrating cells are not MBP-specific memory T cells, thus it is speculated that the up-regulation of the adhesion molecules in the endothelial cells plays a decisive role in the development of immune-mediated diseases of the central nervous system. Therapeutic potency of clinical usage of anti-adhesion molecule antibodies has been explored. (27 Refs.)

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?s vla and 21(w)6

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       1659018  6
          5409  21(W)6
     S3      3  VLA AND 21(W)6
?rd s3

...completed examining records
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?t s4/3/all


 4/3/1     (Item 1 from file: 55)
DIALOG(R)File  55:BIOSIS PREVIEWS(R)
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10801152     BIOSIS Number: 97001152
  VLA-4-dependent adhesion activities of U937 cells and guinea-pig
bronchoalveolar lavage leukocytes
  Monshizadegan D A; Holloway D A; Torrente J M; Yednock T; Fritz L; Sturm
R J
  Wyeth-Ayerst Res., CN 8000, Princeton, NJ 08543, USA
  Agents and Actions 39 (SPEC. CONF. ISSUE). 1993.  C177-C179.
  Full Journal Title:  Agents and Actions
  ISSN: 0065-4299
  Language: ENGLISH
  Print Number: Biological Abstracts Vol. 097 Iss. 001 Ref. 001099
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